Abbreviated Laparotomy and Planned Reoperation for Critically Injured Patients

JON M. BURCH, M.D.,* VICTOR B. ORTIZ, B.A.,* ROBERT J. RICHARDSON, M.D.,*
R. RUSSELL MARTIN, M.D.,† KENNETH L. MATTOX, M.D.,* and GEORGE L. JORDAN, JR., M.D.*

The triad of hypothermia, acidosis, and coagulopathy in critically injured patients is a vicious cycle that, if uninterrupted, is rapidly fatal. During the past 7.5 years, 200 patients were treated with unorthodox techniques to abruptly terminate the laparotomy and break the cycle. One hundred seventy patients (85%) suffered penetrating injuries and 30 (15%) were victims of blunt trauma. The mean Revised Trauma Score, Injury Severity Score, and Trauma Index Severity Score age combination index predicted survival were 5.06%, 33.2%, and 57%, respectively. Resuscitative thoracotomies were performed in 60 (30%) patients. After major sources of hemorrhage were controlled, the following clinical and laboratory mean values were observed: red cell transfusions—22 units, core temperature—32.1 C, and pH—7.09. Techniques to abbreviate the operation included the ligation of enteric injuries in 34 patients, retained vascular clamps in 13, temporary intravascular shunts in four, packing of diffusely bleeding surfaces in 171, and the use of multiple towel clips to close only the skin of the abdominal wall in 178. Patients then were transported to the surgical intensive care unit for vigorous correction of metabolic derangements and coagulopathies. Ninety-eight patients (49%) survived to undergo planned reoperation (mean delay 48.1 hours), and 66 of 98 (67%) survived to leave the hospital. With the exception of intravascular shunts, there were survivors who were treated by each of the unorthodox techniques. Of 102 patients who died before reoperation 68 (67%) did so within 2 hours of the initial procedure. Logistic regression showed that red cell transfusion rate and pH may be helpful in determining when to consider abbreviated laparotomy. The authors conclude that patients with hypothermia, acidosis, and coagulopathy are at high risk for imminent death, and that prompt termination of laparotomy with the use of the above techniques is a rational approach to an apparently hopeless situation.

From the Baylor College of Medicine, The Cora and Webb Mading Department of Surgery, and the Ben Taub General Hospital, Houston, Texas, and the Tripler Army Medical Center,† Hawaii*

CRITICALLY INJURED PATIENTS, such as those suffering from grade V liver injuries or multiple abdominal vascular injuries, may lose blood at the rate of 20 to 40 units per hour while attempts are made to control the hemorrhage. Although anesthesiologists and blood banks may keep pace with this rate of hemorrhage for some time, severe metabolic consequences are inevitable.[1-3] Hypothermia from evaporative and conductive heat loss occurs in spite of warming blankets and blood warmers. The metabolic acidosis of shock is exacerbated by aortic clamping, vasopressors, massive transfusions, and impaired myocardial performance. Coagulopathy due to dilution and hypothermia complete the lethal triad. Each of the above factors reinforces both itself and the other derangements, resulting in an extremely fragile patient who is only minutes away from a fatal arrhythmia. The author's approach to such patients is to break the cycle by abruptly terminating the operation to prevent further heat loss and to transfer the patient to the intensive care unit for rewarming and correction of the coagulopathy. Implicit with this decision is that sufficient time may not be available to treat all injuries. Also, the massive edema of the bowel and retroperitoneum that inevitably occurs in this setting may preclude conventional abdominal closure because the increase in intra-abdominal pressure may cause immediate renal and respiratory failure.[4-11] The purpose of this study is to evaluate the authors' use of unorthodox abdominal closures and other temporary measures to prevent the imminent death of these critically injured patients.

From December 1983 through April 1991, 200 critically injured patients were treated at the Ben Taub General Hospital in Houston, Texas. A variety of unorthodox methods were used to abruptly terminate the initial laparotomy, with the intention of completing the procedure at a planned reoperation.

Data were obtained from patient records and supple-

Presented at the 103rd Annual Scientific Session of the Southern Surgical Association, Hot Springs, Virginia, December 1-4, 1991.

Address reprint requests to Jon M. Burch, M.D., Associate Professor, Baylor College of Medicine, One Baylor Plaza, Houston, TX 77030.

Accepted for publication December 30, 1991.

mented by information from a weekly surgical log. Complete information was available for 163 patients (82%). This rather low figure was primarily due to the frantic activity of the physicians and nurses who treated the patients. The ordering of tests and recording of observations were subordinate to the exigencies of patient care. Percentages were based on available data and are specified where necessary. Patients with incomplete data were excluded from statistical calculations.

There were 183 (92%) men and 17 (8%) women, who ranged in age from 15 to 77 years, with a mean of 31 years. Penetrating trauma was the cause of 170 (85%) injuries, and blunt trauma was responsible for the remaining 30 (15%). The Revised Trauma Score, Injury Severity Score, and Trauma Index Severity Score age combination index (TRISS) predicted survival for patients with penetrating injuries were 5.0%, 32%, and 57%, respectively (median, 6.4%, 26%, and 76%). For blunt trauma, the mean Revised Trauma Score, Injury Severity Score, and TRISS were 5.4%, 41%, and 60%, respectively (median, 6.4%, 41%, and 69%).

Emergency center resuscitation generally followed American College of Surgeons Advanced Trauma Life Support Program (ATLS) guidelines.[12] Sixty patients (30%) required resuscitative thoracotomies either in the emergency center or in the operating room (OR). The median delay from admission to operation was 25 minutes for patients with penetrating injuries and 80 minutes for patients injured by blunt trauma. Median systolic blood pressure on admission was 72 mmHg and was similar for patients with both blunt and penetrating injuries.

In the OR, all patients were explored through midline incisions. Injuries often associated with severe hemorrhage are listed in Table 1. One hundred twenty-five patients suffered major abdominal vascular injuries. In addition, 10 patients suffered major vascular injuries outside the abdominal cavity. Of 102 patients with liver injuries, 15 had juxtahepatic venous injuries (grade V), and 11 required atriocaval shunts.

Physiologic parameters, including pH, core temperature, and systolic blood pressure, were noted at the beginning and end of the initial operation. The lowest values of each were also recorded (Table 2). Intraoperative transfusion and fluid requirements are shown in Table 3. Every attempt was made to warm blood products and crystalloids solution to 37 C before administration. The rate of transfusion of red blood cells ranged from 1.3 to 42.7 U/hour during the operation. The mean rate of red cell transfusion was 11.7 U/hour (median, 10.9), and 20 patients (10%) received red cells at a rate in excess of 20 U/hour. Pharmacologic support of cardiac output was required in 126 patients (63%). Epinephrine was used most often (116 patients); the remaining 10 patients were treated with dopamine.

TABLE 2. *Mean Physiologic Parameters During the Initial Operation*

	Beginning of Operation	Lowest Recorded Value	End of Operation
Systolic blood pressure (mmHg)	102	63*	102*
Core temperature (C)	34.0	32.1	32.7
pH	7.18	7.09	7.19

* Excludes patients who died in the operating room (blood pressure = 0).

On concern of the patient's impending death, several techniques were used to terminate the operation. Laparotomy pads were used to tamponade diffusely bleeding surfaces in 171 patients (86%). The liver was packed in 74 patients (38%), the retroperitoneum in 65 (33%), and the pelvis in 32 (16%). Thirty-four patients with enteric injuries (17%) requiring resection and anastomosis or colostomy were treated by ligating or stapling the intestine on both sides of the injuries (Fig. 1).13 This was done to prevent further contamination of the peritoneal cavity. In most patients, ligation resulted in isolated intestinal segments similar to closed loop bowel obstructions.

Temporary intravascular shunts, secured with snare tourniquets, were used in four patients. All shunts were used for injuries that would have required interposition grafts or end-to-end anastomosis, but sufficient time for the repair was not available. One was used to bridge a defect between the common iliac vein and vena cava, and three were used to bridge defects in the iliac artery.

Aortic clamping was used to support circulation to vital

TABLE 1. *Selected Associated Injuries*

Vascular		Solid Organ	
Inferior vena cava	48	Liver	102
Aorta	21	Pancreas	30
Iliac	66	Kidney	41
Renal	45	Spleen	25
Major splanchnic	50	Pelvic fracture	9
Heart	7		
Other major vascular	21		

TABLE 3. *Mean Transfusion and Fluid Requirements*

	Initial Operation	24-hr Period After Initial Operation	Reoperation
Crystalloid (L)	11.6	5.9	3.3
Packed red cells (units)	22	9.8	4.1
Fresh frozen plasma (units)	4.4	8.8	1.9
Platelets (units)	6.3	10.7	4.2

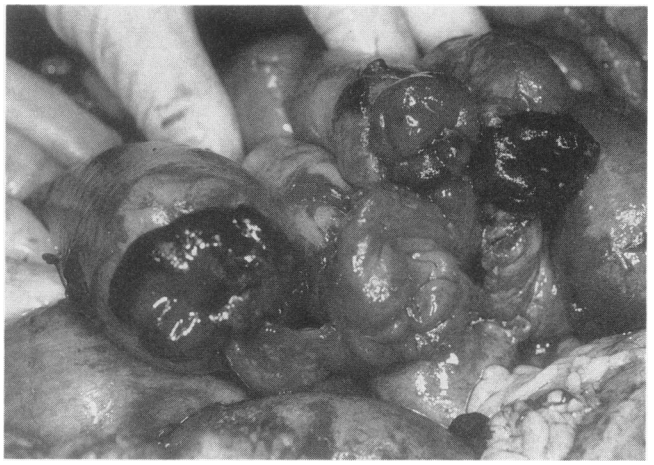

FIG. 1. Appearance of small bowel and colon at reoperation after treatment of original enteric injuries by ligation. The ligatures are still in place.

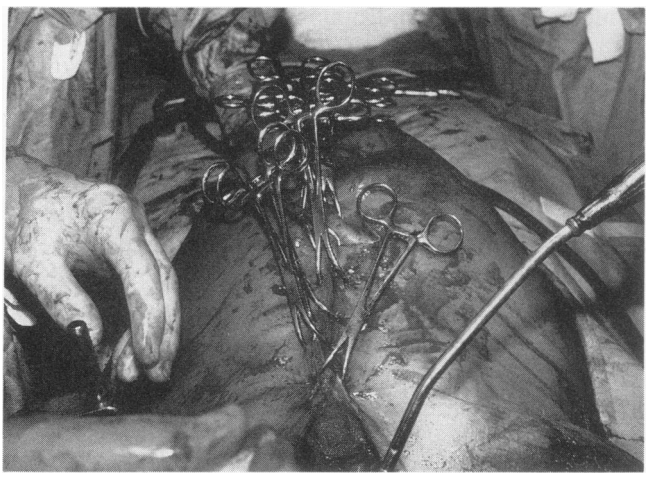

FIG. 3. Towel clip closure. Note the laparotomy pad used to pack the liver at the bottom of the incision.

organs in most patients. In 11 patients, infrarenal aortic clamps could not be removed before abdominal closure without the systolic blood pressure falling to below 50 mmHg, in spite of epinephrine or dopamine. These clamps were brought out through the abdominal incision and gradually released as the patient either improved or died (Fig. 2). Two other patients had intentionally retained vascular clamps on the iliac artery when the clamp controlled associated hemorrhage but the patient's condition precluded further attempts at definitive control.

No patient in this series had a conventional closure of the abdominal incision. The most commonly employed closure was performed with towel clips to approximate only the skin.[4] The clips were placed 1 to 2 cm apart and 1 to 2 cm from the edge of the incision (Figs. 3 and 4). Approximately 25 to 40 towel clips were required, depending on the length of the incision. Towel clip closure was often performed in 60 seconds, although occasionally up to 4 minutes were needed. The clips were covered with a sterile towel and secured with a large plastic adhesive barrier. Towel clip closure was the initial closure in 178 patients (89%). Eleven patients had just the skin of the abdomen closed using a 2 nylon suture. One patient died before abdominal closure; he was included in the series because of treatment with a temporary intravascular shunt.

In 10 patients, massive edema of the intestine and retroperitoneum prohibited approximation of even the skin. Silos were constructed of various rubber or plastic materials after the fashion of techniques used to treat gas-

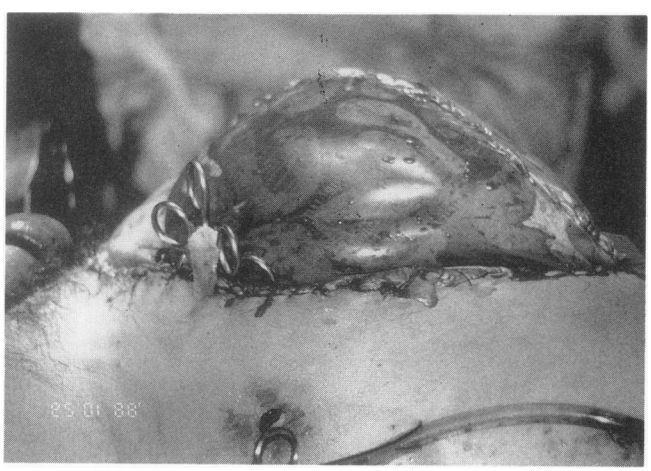

FIG. 2. Retained vascular clamps in a patient with silo closure of abdominal wound. Reprinted with permission from Feliciano DV, Burch JM. Towel clips, silos, and heroic forms of wound closure. *In* Maull KI, ed. Advances in Trauma and Critical Care. Vol. 6. Chicago: Mosby-Year Book, 1991.

FIG. 4. Lateral view of towel clip closure. Only the skin is closed; no attempt is made to include the fascia.

troschisis and omphaloceles in neonates.[14,15] The artificial material was sutured to the skin edges with heavy monofilament suture and tailored to the shape of the incision and the bulging intestine (Fig. 5). Currently, the most popular material is a 3-L Viaflex intravenous fluid bag (Travenol Laboratories, Deerfield, IL) that has been unfolded by cutting the seam and then sterilized (Fig. 6). As many as three or four intravenous bags were sutured together to close large defects.

Results

Survival

Thirty patients (15%) died in the OR. An additional 72 patients (36%) died in the surgical intensive care unit (SICU) before planned reoperation. Figure 7 shows the length of survival for those who died before reoperation. Sixty-eight of 102 patients (67%) died within 2 hours of the end of the initial procedure. For those who reached the SICU alive, vigorous efforts were made to rewarm the patients, correct existing coagulopathies, and optimize oxygen delivery.

Rewarming was accomplished by placing the patients on heating pads (39 C) and covering them with warmed blankets. The return of core temperature to normal required 2 to 8 hours, depending on the degree of hypothermia.

Clotting factors, red cells, and crystalloids administered during the first 24 hours in the SICU (or before reoperation or death if less than 24 hours) are shown in Table 3. In general, more fresh frozen plasma and platelets were given in the SICU than during the initial operation. In contrast, crystalloid infusions were intentionally minimized to prevent further edema of the bowel and associated increases in intra-abdominal pressure.

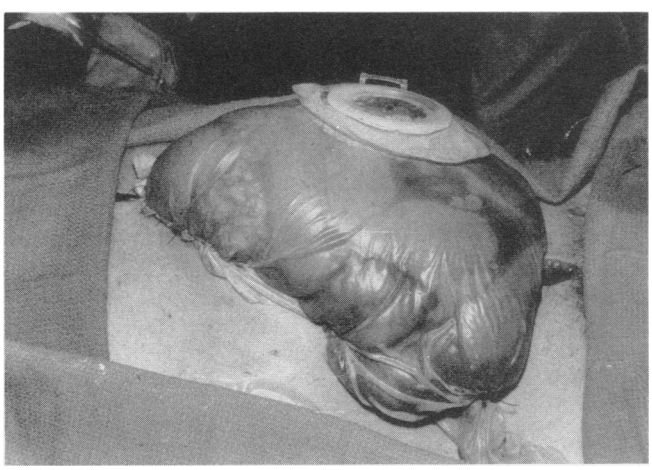

FIG. 5. Large silo constructed of a lightweight waterproof plastic barrier. Note the massive edema of the small bowel. View 24 hr postoperatively, after much of the edema had subsided. Reprinted with permission from Feliciano DV, Burch JM. Towel clips, silos, and heroic forms of wound closure. *In* Maull KI, ed. Advances in Trauma and Critical Care. Vol. 6. Chicago: Mosby-Year Book, 1991.

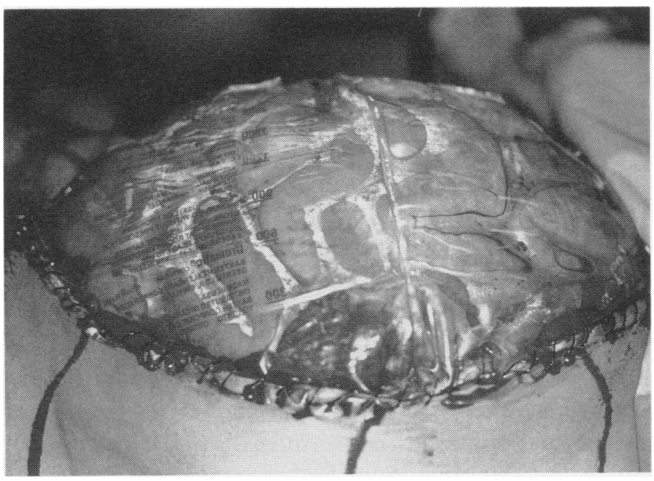

FIG. 6. This silo was constructed from a 3-L urologic irrigation bag, currently the most popular material.

Although all patients had clinically apparent coagulopathies by the end of the initial operation, the results of coagulation studies were seldom available in the operating room. Coagulation studies drawn at the end of the operation or on arrival in the SICU were available for 107 of 144 patients who survived for more than 1 hour in the SICU. The mean prothrombin time was 25.1 seconds (median, 17.5 seconds); the mean partial thromboplastin time was 110 seconds (median, 150 seconds); and the mean platelet count was 97×10^3 cells per mm^3 (median 85×10^3 cells per mm^3). After resuscitation, coagulation studies were available for 79 patients who survived to undergo reoperation. The mean prothrombin time for this group was 15.0 seconds (median, 11.5 seconds); the mean partial thromboplastin time was 46.0 seconds (median,

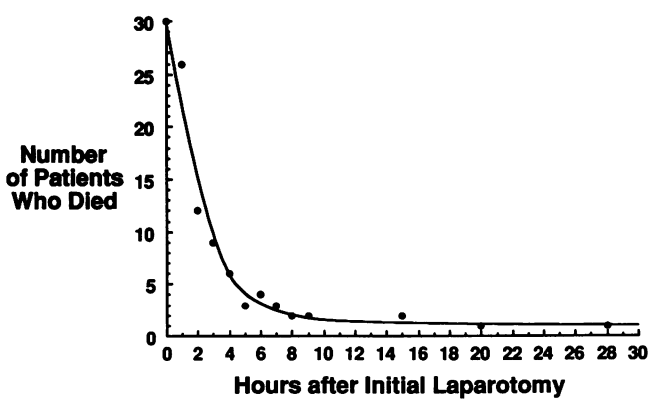

FIG. 7. Length of survival for patients who died before reoperation. Note the large number of patients who die soon after abdominal closure. One patient not included on the figure died 40 hr postoperatively.

35.7 seconds); and the mean platelet count was 120×10^3 cells per mm^3 (median 102×10^3 cells per mm^3).

Dopamine or epinephrine infusions were required to support cardiac output in 81 of 170 patients (48%) who reached the SICU alive. This figure is deceptively low because recording this information was a low nursing priority for patients who died soon after admission. For those who survived to undergo reoperation, dopamine or epinephrine infusions were required in 34 of 98 patients (35%).

Of 98 patients who underwent reoperation, 17 (17%) were operated on because of uncontrolled hemorrhage. The mean time between the end of the initial operation and reoperation was 8.7 hours (median, 10.0 hours; range, 8 to 24 hours) for this group, and only four (24%) survived to leave the hospital. For the remaining 81 patients who underwent planned reoperation, the mean time between procedures was 48.1 hours (median, 48.0; range, 3 to 208), and 62 of 81 patients (77%) survived. The survival rate for all patients undergoing reoperation was 67% (66/98), and for all patients in the series, 33% (66/200). The most common cause of death was exsanguination, which occurred in 110 patients (82%), followed by multiple organ failure in 21 (16%). The causes of death for three remaining patients were adult respiratory distress syndrome, a penetrating head injury, and a pulmonary embolism.

Survival for each technique used to terminate the initial operation is shown in Table 4. With the exception of intravascular shunts, patients survived when treated with each technique. Atriocaval shunts were not considered as adjuncts to terminate the operation. Parenthetically, five of 11 patients (45%) treated with atriocaval shunts survived.

Complications

Eighty-six patients either survived to leave the hospital or lived long enough (>48 hours) to manifest complications. Table 5 lists the significant abdominal complications. Abscesses and enteric fistulas were encountered more often (12% and 8%, respectively) than is usually seen in series of injured patients. Both patients with necrosis of the colon and one of the two with stomal necrosis required epinephrine or dopamine, which may have been a contributing factor.

Patients with organ failure, septic syndrome, and other infections are shown in Table 6. All patients with multiple organ failure had respiratory and renal failure, and many in this category also had hepatic, cardiac, or hematologic failure. The term septic syndrome is used to describe patients with the clinical signs of sepsis (fever, hypotension, diaphoresis, elevated white blood cell count, and decreased systemic vascular resistance) but who do not have an apparent focus of infection. Survival for these patients was particularly poor (one of nine, 11%).

There were 13 serious wound complications, including dehiscence or evisceration in 6 patients, necrotizing infections in 4, large hernias in 2, and necrosis of the skin and subcutaneous tissue in 1. All patients but three were initially treated with towel clip closure. Two were treated with silos, which disrupted and resulted in evisceration. The remaining patient suffered a wound infection; his abdomen was originally closed with heavy nylon suture.

Six patients had serious vascular complications. Two required above-knee amputations; both had intentionally retained vascular clamps, one on the aorta and the other

TABLE 4. *Survival According to Technique*

Technique	No. of Patients	Survivors No. (%)
TCC	178	53 (30)
Nylon	11	8 (73)
Silo	10	5 (50)
Bowel ligation	34	7 (21)
Temporary shunt	4	0 (0)
Retained clamp	13	1 (7.7)
Packed	171	57 (33)

TCC, towel clip closure.

TABLE 5. *Abdominal Complications (N = 86 [66])**

Complication	No. of Patients
Abscess	10 (8)
Fistula	7 (5)
Peritonitis	1 (1)
Small bowel obstruction	3 (3)
Colonic necrosis	2 (1)
Stomal necrosis	2 (1)
Pseudocyst	1 (1)
Urinoma	1 (1)

* Numbers in parentheses indicate survivors.

TABLE 6. *Organ Failure, Septic Syndrome, and Infectious Complications (N = 86 [66])**

Complication	No. of Patients
MOF	25 (3)
ARDS	8 (7)
ARF	2 (2)
Septic syndrome	9 (1)
Pneumonia	10 (7)
Empyema	3 (3)
Catheter sepsis	2 (1)
Urinary tract infection	1 (1)

* Numbers in parentheses indicate survivors.
MOF, multiple organ failure; ARDS, adult respiratory distress syndrome; ARF, acute renal failure.

on the iliac artery. Two patients had thrombosis of the common iliac artery; one had a temporary shunt placed in the vena cava, which thrombosed. The resultant increase in venous pressure appeared to compromise an iliac artery anastomosis. The other had ligation of the vena cava, which was followed by arterial thrombosis. Two of these patients also developed compartment syndromes, one with the retained aortic clamp and the other with ligated vena cava. One patient developed gangrene of the toes related to a high-dose epinephrine infusion that was started in the OR and continued in the SICU. One patient who died of a pulmonary embolus was treated with a retained aortic clamp. He was recovering without serious complications, but as he began to ambulate on the 9th postoperative day, the embolus occurred.

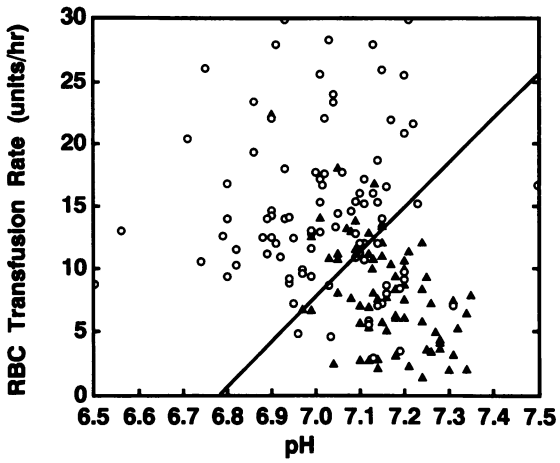

FIG. 8. Scatter plot of pH and red cell transfusion rate. Triangles represent survival greater than 48 hr. Circles represent death within 48 hr. Patients plotted above the diagonal discriminant line are at high risk for death within 48 hr of initial operation. See text.

Discussion

The concept of planned reoperation for trauma patients is not new. Authors from this institution as well as others have recognized the wisdom of packing a diffusely bleeding liver, retroperitoneum, or pelvis and returning at a later date.[5,16,17] The unusual aspect of this series was the surgeon's recognition of the patient's imminent death and instituting measures to terminate the procedure as fast as possible. That the authors were able to predict which patients would die is supported by Figure 7. Of 102 patients who died before reoperation, 56 (55%) died within 1 hour of abdominal closure, and 68 (67%) died within 2 hours.

There are several indicators available to the operating team that may be helpful in predicting impending death. These include cardiac rhythm, red cell transfusions, blood pressure, core temperature, pH, coagulopathy, and various injury severity scoring systems. Ventricular arrhythmias occur in virtually all patients who die in the OR or shortly thereafter. They are characterized by wide QRS complexes, ectopy, bradycardia, and electromechanical dissociation. These arrhythmias are often refractory to antiarrhythmic drugs and cardioversion. In this series, only two of 67 (3.0%) patients who suffered a cardiac arrest in the OR were successfully resuscitated. Although ventricular arrhythmias are excellent predictors of imminent death, it is usually too late to intervene. The presence of coagulopathy is a subjective observation and does not predict imminent death. Although objective, the injury severity indices are only predictive of eventual death.

To evaluate the surgeon's ability to predict impending death, a regression analysis of available intraoperative information was performed. Death within 2 hours of abdominal closure was selected as an end point. The two factors that emerged as independent and most predictive were the rate of red cell transfusion (units per hour) and pH ($p < 0.0001$). Figure 8 is a scatter plot of pH, the rate of red cell transfusion, and 48-hour survival for 163 patients with complete data. The diagonal discriminant line was derived by logistic regression and has the following equation: transfusion rate = 35.7(pH) − 242. Patients to the left and above the line are considered at high risk for imminent death, those to the right and below, low risk. The accuracy of this discriminant (risk of death within 48 hours = 0.57, 77% correct identifications) must be tested in a prospective study to be proven. In lieu of such a study, it seems reasonable to consider abbreviated laparotomy when a patient's transfusion rate and pH approach the discriminant.

Although the triad of hypothermia, acidosis, and coagulopathy has been recognized as a lethal combination, greater emphasis has been placed on the individual elements.[18–21] In a recent review of hypothermia and coagulopathies, Patt et al.[22] noted three mechanisms by which hypothermia interfered with hemostasis: the inhibition of the temperature-dependent enzyme-activated coagulation cascades, increased fibrinolytic activity, and reversible platelet dysfunction. Hypothermia also has profound effects on the heart and circulation, which result in hypotension, bradycardia, ventricular irritability, and metabolic acidosis.[23] It is not surprising that hypothermia has been well established as an accurate predictor of death in trauma patients.[24–26]

Attempts to combat hypothermia in trauma patients have centered around devices to warm blood and fluids for rapid administration.[1,26,27] Although these machines may be capable of transfusion rates of 1.6 L/minute, the logistics of preparing the machine at a moment's notice, procuring the blood, and running the device are formidable. Even if isothermic infusions are maintained, ra-

diant, conductive, and evaporative heat loss in the cold, dry OR environment go unchecked. The authors believe that eliminating these sources of heat loss by closing the abdominal incision and covering the patient with warmed dry blankets is a practical if not critical adjunct.

In addition to hypothermia, other causes of coagulopathy in this setting include dilution and consumption.[1,3,28] All patients in the present series received red cell transfusions in the form of packed red cells that contain virtually no clotting factors or platelets. Only the residual plasma proteins in the patient's circulation and those produced by the liver during the operation can contribute to coagulation. Transfusion of two or more blood volumes of saline and packed red cells has been shown to decrease the level of several clotting factors to 15% of normal.[3] Platelets are lost by disseminated intravascular coagulation in addition to dilution.[1,3,28]

Most authors recommend that the use of fresh frozen plasma and platelets be based on coagulation studies and platelet counts.[1,3] Unfortunately, during the time the studies are performed, another 5 to 10 L of blood may be lost during the operation, and the patient's coagulation profile will bear little resemblance to that which was present when the tests were drawn. It is therefore necessary to replace clotting factors empirically.

Metabolic acidosis affects both the circulatory system and coagulation. Acidosis has been shown to decrease myocardial contractility and cardiac output.[29,30] Although it is known that acidosis decreases coagulation time, there is evidence that it may also trigger diffuse intravascular coagulation and cause a consumptive coagulopathy.[31] The importance of metabolic acidosis has been recognized in other clinical studies, but documentation has been scarce.[20,32] In the present study, acidosis was more predictive of impending death than hypothermia.

In addition to the above metabolic problems, the surgeons must also address the technical problem of closing the abdomen. The edema that occurs in the intestine and retroperitoneum is due to an increase in interstitial fluid and may be related to low colloid osmotic pressure.[33] This edema may be so great that abdominal closure is impossible (Fig. 5). In less dramatic circumstances, closure of even the skin may produce intra-abdominal pressures of 50 mmHg or more, as occurred in several patients in this series.

In laboratory models, intra-abdominal pressure of 30 to 40 mmHg has been shown to decrease cardiac output, redistribute blood flow from the abdomen to the thorax, cause ventilation/perfusion mismatches in the lungs, and impair renal function.[6–8,34] Clinical studies have emphasized increased intra-abdominal pressure as a cause of acute renal failure.[9–11] The cause of renal failure is not certain, but is probably related to compression of the kidney or renal veins. Decompression usually leads to a prompt return of renal function.[9–11]

Richardson and Trinkle[6] observed that increased intra-abdominal pressure in dogs caused an increase in end inspiratory pressure, a redistribution of blood flow in the lungs, ventilation/perfusion mismatches, and hypoxia. Three patients in this series had peak inspiratory pressures in excess of 80 cmH_2O after towel clip closure. None could be effectively ventilated, and all required removal of towel clips and the construction of silos. Apprahamian et al.[5] recently reported the utility of silos to prevent lethal increases in IAP.

In conclusion, the triad of hypothermia, coagulopathy, and acidosis is a vicious circle that heralds imminent death. The use of towel clip closure, silos, abdominal packs, ligation of enteric injuries, and other techniques permit the abrupt termination of the operation so that resuscitation can be accomplished. Closing the abdomen prevents further heat loss and aids in tamponade of diffuse bleeding. The use of pH and red cell transfusion rate may be helpful in determining when to abandon the initial operation. For patients who stabilize and undergo planned reoperation, the survival is remarkably good.

Acknowledgments

The authors thank Kenneth Hess, M.S., of the Sakowitz Computer Laboratory, the Cora and Webb Mading Department of Surgery, Baylor College of Medicine for statistical analyses, and a special note of appreciation is given to Marian Torres for her assistance in data collection and manuscript preparation. The authors also thank the Baylor College of Medicine general surgery residents for their perseverance and ingenuity in the treatment of these patients.

References

1. Collins JA. Problems Associated with the massive transfusion of stored blood. Surgery 1974; 75:274–295.
2. Phillips TF, Soulier G, Wilson RF. Outcome of massive transfusion exceeding two blood volumes in trauma and emergency surgery. J Trauma 1987; 27:903–910.
3. Maier RV. The consequences of massive blood tranfusion. Surg Rounds 1984; 7:57–84.
4. Feliciano DV, Burch JM. Towel clips, silos, and heroic forms of wound closure. *In* Maull KI, ed. Advances in Trauma and Critical Care. Volume 6. Chicago: Mosby Year Book, 1991.
5. Aprahamian C, Wittman DH, Bergstein JM, Quebbeman EJ. Temporary abdominal closure (TAC) for planned relaparotomy (etapenlavage) in trauma. J Trauma 1990; 30:719–723.
6. Richardson JD, Trinkle JK. Hemodynamic and respiratory alterations with increased intra-abdominal pressure. J Surg Res 1976; 20:401–404.
7. Robotham JL, Wise RA, Bromberger-Barnea B. Effects of changes in abdominal pressure on left ventricular performance and regional blood flow. Crit Care Med 1985; 13:803–809.
8. Barnes GE, Laine GA, Gian PY, et al. Cardiovascular responses to elevation of intra-abdominal hydrostatic pressure. Am J Physiol 1985; 248:208–213.
9. Richards WO, Scovill W, Shin B, Reed W. Acute renal failure associated with increased intra-abdominal pressure. Ann Surg 1983; 197:183–187.

10. Kron IL, Harman PK, Nolan SP. The measurement of intra-abdominal pressure as a criterion for abdominal reexploration. Ann Surg 1984; 199:28–30.
11. Daly RC, Mucha PJ, Farnell MB. Abdominal reexploration for increased intraabdominal pressure and acute oliguric renal failure. Cont Surg 1989; 35:11–18.
12. Advanced Trauma Life Support Course Book. Chicago: American College of Surgeons, 1990.
13. Burch JM, Martin RR, Richardson RJ, et al. Evolution of the treatment of the injured colon in the 1980's. Arch Surg 1991; 126: 979–984.
14. Schuster SR. A new method for the staged repair of large omphaloceles. Surg Gynecol Obstet 1967; 125:837–850.
15. Allen RG, Wrenn EL Jr. Silon as a sac in the treatment of omphalocele and gastroschisis. J Pediatr Surg 1969; 4:3–8.
16. Feliciano DV, Mattox KL, Burch JM. Packing for control of hepatic hemorrhage. J Trauma 1986; 26:738–743.
17. Cue GL, Cryer HG, Miller FB, et al. Packing and planned reoperation for hepatic and retroperitoneal hemorrhage: critical refinements of a useful technique. J Trauma 1990; 30:1007–1011.
18. Feliciano DV, Pachter HL. Hepatic trauma revisited. Curr Probl Surg 1989; 26:453–524.
19. Ferrara A, MacArthur JD, Wright HK, et al. Hypothermia and acidosis worsen coagulopathy in the patient requiring massive transfusion. Am J Surg 1990; 160:515–518.
20. Dunham CM, Belzberg H, Lyles R, et al. The rapid infusion system: a superior method for the resuscitation of hypovolemic trauma patients. Resuscitation 1991; 21:207–227.
21. Burch JM, Feliciano DV, Mattox KL. The atriocaval shunt: facts and fiction. Ann Surg 1988; 207:555–568.
22. Patt A, McCroskey BL, Moore EE. Hypothermia-induced coagulopathies in trauma. Surg Clin North Am 1988; 68:775–785.
23. Reuler JB. Hypothermia: pathophysiology, clinical settings, and management. Ann Intern Med 1978; 89:519–527.
24. Luna GK, Maier RV, Pavlin EG, et al. Incidence and effect of hypothermia in seriously injured patients. J Trauma 1987; 27:1014–1018.
25. Jurkovich GJ, Greiser WB, Luterman A, Curreri PW. Hypothermia in trauma victims: an ominous predictor of survival. J Trauma 1987; 27:1019–1024.
26. Gregory JS, Flancbaum L, Townsend MC, et al. Incidence and timing of hypothermia in trauma patients undergoing operations. J Trauma 1991; 31:795–800.
27. Falcone RE, Fried SJ, Zeeb P, Satiani B. Rapid volume replacement with warmed blood and fluids. J Vasc Surg 1989; 9:964–969.
28. Counts RB, Haisch C, Simon TL, et al. Hemostasis in massively transfused trauma patients. Ann Surg 1979; 190:91–99.
29. Wildenthal K, Mierzwiak DS, Myers RW, Mitchell JH. Effects of acute lactic acidosis on left ventricular performance. Am J Physiol 1968; 214:1352–1359.
30. Yudkin J, Cohen RD, Slack B. The haemodynamic effects of metabolic acidosis in the rat. Clin Sci Mol Med 1976; 50:177–184.
31. Hardway RM. Influence of vasoconstrictors and vasodilators on disseminated intravascular coagulation in irreversible hemorrhage shock. Surg Gynecol Obstet 1964; 119:1053.
32. Ferrara A, MacArthur JD, Wright HK, et al. Hypothermia and acidosis worsen coagulopathy in the patient requiring massive transfusion. Am J Surg 1990; 160:515–518.
33. Bock JC, Barker BC, Clinton AG, et al. Post-traumatic changes in, and effect of colloid osmotic pressure on the distribution of body water. Ann Surg 1989; 210:395–405.
34. Kashtan J, Green JF, Parsons EQ, Holcroft JW. Hemodynamic effects of increased abdominal pressure. J Surg Res 1981; 30:249–255.

Discussion

Dr. Timothy C. Fabian (Memphis, Tennessee): Drs. Bland and Jones, Members and Guests, I would like to congratulate the authors on application of some very innovative and novel approaches to salvage an awfully large group of catastrophically injured patients. We use some but not all of these techniques in Memphis. I think, certainly, one of the most beneficial I have observed is the rapid towel clip closure, and I recommend it to all of you in the audience. It can be very helpful and save lots of time.

I have the following series of questions. You alluded to this a couple of times, but I would like for you to expand on it a bit. With your TRISS methodology, you were supposed to have 57% survivals with penetrating trauma, and you actually only had 33%. I suspect this may be a limitation and a fault of quality assurance measures used for that analysis, but it bothers me that in the future all of us are going to be looking at such criteria for trauma center evaluation, because it is being proposed by many different groups, including the Joint Commission on the Accreditation of Hospitals and others.

In your manuscript, you reported resuscitative thoracotomy in the emergency room or operating room in 30%. Does this contribute to hypothermia? You are opening up another celomic cavity. We rarely do this, with the exception of patients with cardiac injuries, and I suspect that this does cause some problems. What is the mortality rate in those 30 patients? Your rate of transfusions was 1 U/hour to 43 in your manuscript. I would like to know the bell-shaped curve of those that were not bleeding very much. Why were they so catastrophically injured? What sort of unique circumstances were these patients?

Turning to the method of major vascular management, you had four shunts placed, one in the inferior vena cava and three in the ileac arteries. I would like to know the results of those specific procedures. No matter how coagulopathic these patients were, I suspect that they all thrombosed their shunts. And what was the outcome?

Additionally, your 11 aortic clamps and two ileac clamps that were left, what was the outcome of these? The incidence of compartment syndromes in these kinds of patients at our institution is relatively high, and we are very aggressive about putting catheters in for compartment pressure measurement after operation. What is your experience with compartment syndromes and loss of compartments and amputations in these patients?

Very importantly, an abdominal wall defect results from using silos that you create with plastic IV bags. Our experience has been that we can almost never get those bellies closed, and we end up putting split-thickness skin grafts on granulation tissue with definitive reconstruction at 8 to 12 months of large ventral hernias. How do you manage them in Houston? And you also mentioned that you do not use clotting studies for fresh frozen plasma, which I think none of us do in the operating room for these, again, seriously injured patients. What is the Houston formula for replacement of platelets and fresh frozen plasma when dealing with massive transfusions and coagulopathy? Thank you very much.

Dr. R. Neal Garrison (Louisville, Kentucky): Vice President Bland, Secretary Jones, Members and Guests, Dr. Burch and colleagues are to be congratulated for an in-depth clinical study of a very complex problem, the operative management of a patient with severe hemorrhagic shock. There is much for the readers of this manuscript to consider and possibly adapt in their practice and care for the patients. And I recommend the manuscript to you. The real question for the recommended technique is when is the best time to implement such a strategy, to hasten the procedure, and to transfer the care to the intensive care unit setting *versus* continued attempts to control bleeding in the operating room.

I have two questions for the authors. You use only two variables in predicting outcome, the number of transfusions given to the patient and the systemic patient pH. What other variables were considered for your predicted equation? There are several that come to mind and have been used to manage the severity of injury or hemorrhage, such as the degree or length of time of blood pressure reduction or systemic oxygen utilization as measured by mixed venous oxygen saturation.

Secondly — and this is along similar lines of one of Dr. Fabian's